An Expanding Serpiginous Plaque of the Lower Extremity

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Case Report

A 51-year-old man with no past medical history presented to the outpatient dermatology clinic with a complaint of severe pruritus of his right lower leg for one week's duration. He reported no history of any skin problems in the past, including any history of atopy. He denied any recent contacts, but admitted to recent travel to Mexico where he swam in ocean water and walked barefoot on the beach several times prior to experiencing any symptoms. The patient described the initial lesion as a "small pink bump" that continued to enlarge and become more itchy. He could not recall any "bites" or "stings," and denied systemic symptoms, such as headache, fevers, or chills. Physical examination revealed an erythematous and scaly serpiginous plaque approximately 6cm in length on the right shin (Figure 1). A punch biopsy of the skin was sent for histological analysis (Figures 2 and 3). Special staining for CD30 was also performed (Figure 4).

Diagnosis

Cutaneous larva migrans (CLM)

Microscopic Findings and Clinical Course

The skin biopsy histological anlysis revealed a superficial and deep perivascular dermatitis predominantly composed of lymphocytes (Figure 2). There were also eosinophils present, extending both around blood vessels and between collagen bundles (Figure 3). There was no evidence of tunneling larvae in the epidermis or dermoepidermal junction. Similar patterns can be seen in arthropod assault reactions, papular urticaria, and dermatoses in which a precise source of antigenic stimulation is not clearly evident. Similar changes have been described in response to other types of ectoparasites, such as helminths. Immunohistochemical staining demonstrated scattered CD30-positive cells most consistent with a reactive phenomenon (Figure 4). Based on the clinical history, lesional morphology, and histological findings, a diagnosis of CLM was made. The patient was subsequently treated with ivermectin 12mg orally in a single dose with complete resolution of clincal findings within one week (Figure 5).

Discussion

CLM, also known as creeping eruption, ground itch, or dew itch, is the most commonly acquired tropical dermatosis. The clinically characteristic serpiginous nature of the skin plaque is caused by the transdermal penetration and ensuing migration of the animal hookworm larvae. Intestinal nematode larvae from domestic dogs (*Ancylostoma caninum*) and/or cats (*Ancylostoma braziliense*) are usually the culprits.[1,2] Ground itch refers to a similar eruption caused by a human species of hookworm (*Ancylostoma duodenale* and *Necator americanus*). Unfortunately, autoinfection is possible with this variant, even up to years after the initial infection.[1–3]

CLM is most commonly seen in warm, tropical climates, such as the southeastern United States, Central and South America, and Africa, although it can be seen worldwide, especially with the increased ease of international travel. The typical description is that of a pruritic, erythematous, edematous papule, plaque, or vesicle that slowly expands in a serpiginous configuration. Infection is most

commonly seen on the feet or buttocks after contact with sand or soil contaminated with animal feces. The larvae migrate roughly 1 to 2cm per day, which is the main cause of intense pruritus and the telltale visible tracts within the skin.[2,3] This is in contrast to larva currens, the pathognomonic cutaneous eruption associated with strongyloidiasis, which evolves rapidly at a rate of 5 to 15cm per hour.[4]

Fortunately, the disease is generally self-limited as the larvae lack lytic collagenases necessary to penetrate through the epidermal basement membrane.[3] Additionally, the animal hookworm larvae are unable to complete their life cycles within a human host, as it is not a natural reservoir for the organism. Thus, the larvae usually die within 2 to 8 weeks after initial skin penetration, resulting in complete skin resolution.[3] It is uncommon for the skin disease to persist if left untreated. In rare cases in which the larvae migrate beyond the epidermal basement membrane, systemic signs, including peripheral eosinophilia (Loeffler's syndrome), migratory pulmonary infiltrates, and increased immunoglobulin E levels, can be seen, but will resolve with systemic therapy.[5,6] It is not uncommon for secondarily impetiginized lesions to occur due to the intense pruritus and subsequent scratching.

The diagnosis of CLM is generally made by clinical exposure history and typical serpiginous tracts on physical examination. It is uncommon to demonstrate the hookworm within skin biopsy, but the larva may occasionally be visualized within the epidermis.

Treatment for CLM includes oral antiparasitic therapy with albendazole 400 to 800mg orally for 3 to 5 days in adults; however, a single dose of

Figure 1. Serpigenous, pink, scaly plaque on the right shin

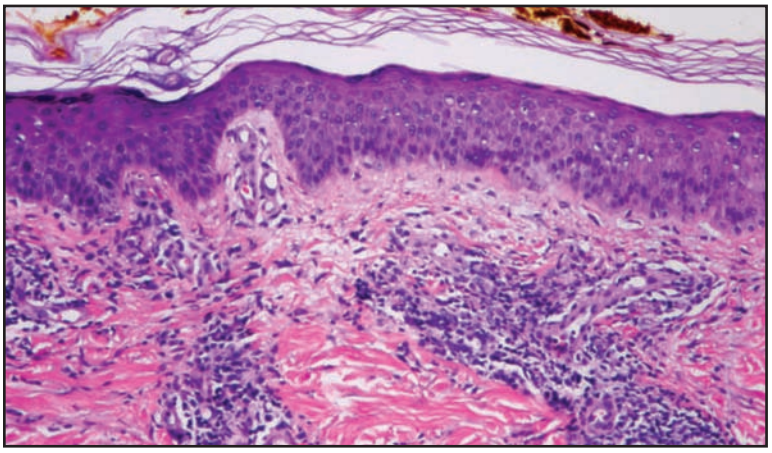

Figure 2. Collections of mononuclear cells surrounding vessels in the superficial and deep plexus (hematoxylin and eosin, 10x)

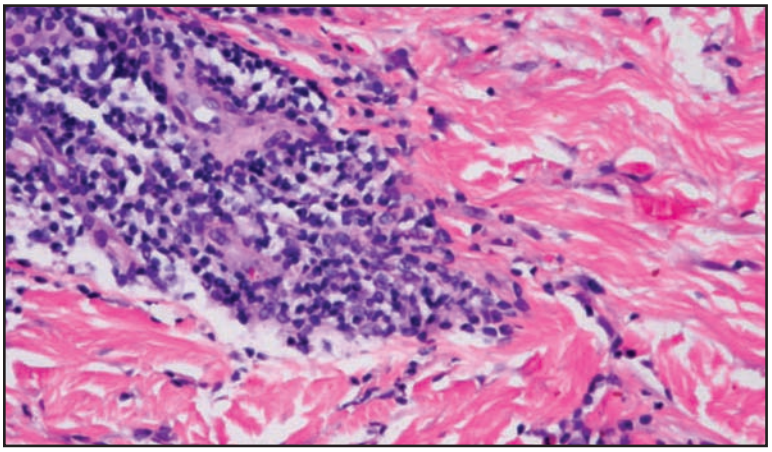

Figure 3. There are eosinophils that extend both around the vessels and between collagen bundles (hematoxylin and eosin, 20x)

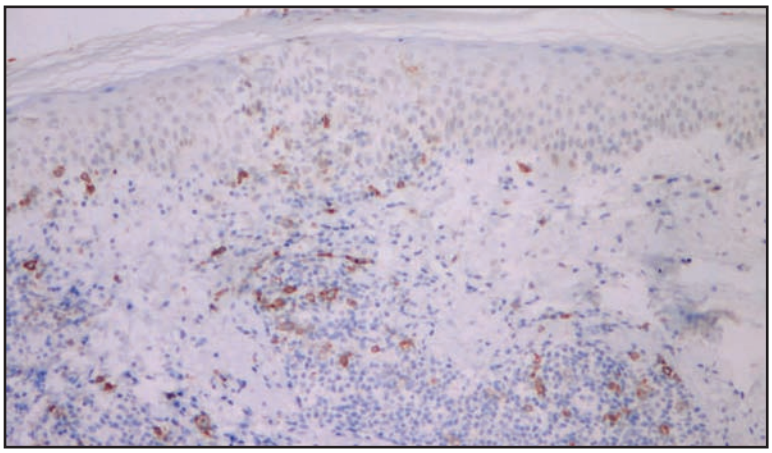

Figure 4. Scattered staining for CD30 (hematoxylin and eosin, 10x)

Figure 5. Plaque clearance after treatment with oral ivermectin

ivermectin 12mg orally is also highly effective.[6,7] Weight-based dosing with the same agents are also effective for treating children.[5,7] Topical therapy with thiabendazole 15% cream is available for localized disease in which oral therapy is not recommend or contraindicated, such as in pregnant or nursing patients. However, this medication requires application 2 to 3 times per day for approximately two weeks and thus, is a less favorable option.[5,6]

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